Remarks

Reconsideration of this Application is respectfully requested.

Upon entry of the foregoing amendment, claims 35, 36, 38, 42, 43, 45-48, and renumbered claim 51 are pending in the application. Claim 43 has been amended. Support for the amended claim can be found at least at page 13, line 24 (Figure 6) and page 19, lines 16-21. These changes are believed to introduce no new matter, and their entry is respectfully requested.

Based on the above amendment and the following remarks, Applicants respectfully request that the Examiner reconsider all outstanding objections and rejections and that they be withdrawn.

I. Miscellaneous

Applicants wish to thank the Examiner for withdrawing rejections of claims 35, 42-43, 47-48, and 51 under 35 U.S.C. § 102(a) and rejection of claims 35, 45, and 46 under 35 U.S.C. § 103(a) in light of Applicant's Declaration submitted under 37 C.F.R. 1.131.

The Examiner also noticed that the references submitted August 21, 2001 and cited in Paper No. 6 were not available for consideration at the time of examination. The undersigned has contacted Maura Barreto at the U.S.P.T.O. (703-305-5727) and has been informed that the documents cited in the 1449 form can be found in the SPE's office.

Therefore, Applicants are resubmitting the 1449 form previously filed in the U.S.P.T.O on August 21, 2001 and again request the Examiner to indicate consideration of documents by signing the 1449 form.

II. Rejections under 35 U.S.C. § 112 (Written Description)

The examiner has rejected claim 43 under 35 U.S.C. § 112, first paragraph (Paper No.10, pg. 3), on the grounds that the claim allegedly contains "subject matter which was not described in the specification in such as way as to reasonably convey to one skilled in the relevant art that the inventor(s), at the time the application was filed, had possession of the claimed." Applicants respectfully traverse this ground for rejection.

Solely in an effort to expedite prosecution, however, and without acquiescence in the propriety of the rejection, applicants have amended claim 43 to recite DNA sequences encoding at least 400 amino acids of SEQ ID NO: 16. Thus, Applicants respectfully request that the Examiner reconsider and withdraw the rejections.

III. Rejections under 35 U.S.C. § 112 (Enablement)

The Examiner has maintained his rejection of Claims 35, 36, 38, 43, 45-46, and 48 for not being enabled by the specifications. Applicants respectfully traverse the rejection.

A. Claim 43

In the Office Action dated November 26, 2001 at page 7, the Examiner rejected claim 43 based on the arguement that the claim encompassed "a DNA sequence encoding any JAK kinase peptide, not necessarily limited to a JAK 1, JAK 2, or JAK 3 kinase peptide, derived from any animal species wherein said peptide has cytokine receptor binding activity...." The claim has been amended as stated above to encompass DNA sequences encoding "at least 400 amino acids of a JAK3 peptide of SEQ ID NO: 16; wherein said peptide binds a receptor for a cytokine selected from a group" as stated in amended claim 43. Accordingly, Applicants respectfully request that the Examiner reconsider and withdraw the objection.

B. Claims 35, 36, 38, 45-46, and 48

The Examiner appears to base his rejection of the remaining claims on the following arguments: i.e. a "lack of guidance provided by the instant specification" and "unpredictability of the art on protein/peptide folding and tertiary structure prediction" resulting in "undue experimentation for one skilled in the art to make and use the broadly claimed invention". (Paper No. 10, pg. 10). Applicants respectfully disagree.

By "lack of guidance" the Examiner argues that despite the limitation of claim 35 to "a DNA sequence encoding at least 400 amino acids of a Jak3 kinase peptide of SEQ ID NO: 16, wherein said peptide has Jak kinase activity and undergoes tyrosine phosphorylation by at least one cytokine...," selected from a group of cytokines, the specification fails to teach specifically which JAK3 kinase peptides derived from SEQ ID

NO: 16, containing which domains or critical regions would be required for JAK kinase activity and tyrosine phosphorylation by the group of cytokines.

Applicants wish to point out that information such as critical kinase regions or domains of a protein with kinase activity are well known in the art and were used in developing the present invention (Page 49, lines 9-13). Furthermore, figure 6 of the specifications provides an amino acid sequence alignment between murine JAK 1, 2, and 3 and human Tyk2. (Page 13, line 24). Positions in the proteins in which three or more amino acids are identical are noted. The skilled artisan would recognize that conserved regions between two or more proteins having similar biological activity are indicative of regions important to that biological activity. Here again, the skilled artisan would know to minimize or avoid making insertion, substitutions, or deletions of amino acid residues within these regions when attempting to produce a variant protein which retains JAK3 kinase and/or receptor binding activity which is "at least 400 amino acids" of SEQ ID NO:16.

Applicants also reiterate, from a previous reply dated August 28, 2001, that the "at least 400 amino acid" limitation represents a significant portion of the complete sequence and one of skill in the art, in light of what is taught in the specification, should have no difficulty in selecting the appropriate portion of the molecule to use in the claimed invention. .

Furthermore, even assuming *arguendo,* that not all specific embodiments of the claimed invention might be enabled. Applicants have still provided sufficient teaching to meet the requirements under 35 U.S.C. § 112, first paragraph. Applicants have taught the ability to make a peptide (Page 20, line 4 - Page 27, line 11), and then test for JAK

kinase activity (Page 24, line 21 - Page 25, line 29), and tyrosine phosphorylation (Page 19, lines 28-31 - Page 20, lines 1-4) is taught. Thus, the skilled artisan would not only be aware of critical kinase regions and domains from information in the art and specification, but could also test JAK3 peptides for activity and phosphorylation by routine experimentation as taught in the specification.

The Examiner also argues that the art is unpredictable with respect to predicting protein folding and tertiary structure; therefore undue experimentation would be required to practice the claimed invention. However, it is unclear how the prediction of JAK3 tertiary structure is required to practice the claimed invention. Again, the skilled artisan would only require the ability to make a peptide and then test for JAK kinase activity and/or phosphorylation which are all routine assays to one skilled in the art and are taught in the specifications. In any event, a minimal level of permitted unpredictability does not result in undue experimentation.

In view of the functionally significant regions in the art and specifications and the fact that routine assays for determining kinase and receptor binding activity of JAK3 are described in the specifications, it would not require undue experimentation for one of ordinary skill in the art to make and/or use nucleic acid variants encoding "at least 400 amino acids of JAK3 SEQ ID NO:16" that have JAK kinase activity and undergo tyrosine phosphorylation. Therefore, the claimed invention is enabled and Applicants respectfully request that the Examiner reconsider and withdraw the rejection of claims 35, 36, 38, 45-46 and 48.

IV. Rejections under 35 U.S.C. § 102(b)

Claim 43 was rejected under 35 U.S.C. § 102(b) as allegedly being anticipated by Wilks *et al* (Mol. Cell. Bio. 11: 2057-2065, 1991). The Examiner argues that Wilkes *et al* teaches the isolation of the full length human JAK1 kinase cDNA. Claim 43 has been amended to read "at least 400 amino acids of a JAK3 peptide of SEQ ID NO; 16". Therefore, the rejection is now moot.

V. Rejection of Claims 35-36, 42, 45-47, and 51 Obviousness-Type Double Patenting

In the Office Action at page 13, the Examiner rejected claims 35-36, 42, 45-47, and 51 under the judicially created doctrine of obviousness-type double patenting as being unpatentable over claims 1-8 of US Patent No. 6,136,595. Applicants respectfully traverse this rejection, however, request that it be held in abeyance until claims are otherwise in condition for allowance.

Conclusion

All of the stated grounds of objection and rejection have been properly traversed, accommodated, or rendered moot. Applicants therefore respectfully request that the Examiner reconsider all presently outstanding objections and rejections and that they be withdrawn. Applicants believe that a full and complete reply has been made to the outstanding Office Action and, as such, the present application is in condition for allowance. If the Examiner believes, for any reason, that personal communication will

expedite prosecution of this application, the Examiner is invited to telephone the undersigned at the number provided.

Prompt and favorable consideration of this Amendment and Reply is respectfully requested.

Respectfully submitted,

STERNE, KESSLER, GOLDSTEIN & FOX P.L.L.C.

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Attorney for Applicants
Registration No. 35,086

Date: Feb. 26, 2002

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Version with markings to show changes made

Paragraph beginning on page 128, line 10:

Following addition of CNTF, a receptor complex forms that consists of CNTF, CNTFRα, gp130, and LIFRβ. Immunoprecipitation (IP) of the receptor complex with antibodies against LIFRβ [(Figure 10)] or gp130 (not shown) following cell lysis in the detergent Brij 96 results in the co-purification of a 130 kDa protein that is tyrosine phosphorylated. LIF and OSM, which also bind to and heterodimerize gp130 and LIFRβ (Gearing *et al.*, *Science 260*:1434-1437 (1992); Baumann *et al.*, *J. Biol. Chem. 268*:8414-8417 (1993); Davis *et al.*, *Science 250*:1805-1808 (1993)), also show association and tyrosine phosphorylation of a protein with an identical appearance [(Figure 10)]. The purified receptor complex also shows associated protein tyrosine kinase activity *in vitro* giving rise to tyrosine phosphorylation of both gp130 and LIFRβ, as well as the associated 130 kDa protein. Tyrosine kinase activity is also associated with LIFRβ in the absence of CNTF, although the 130 kDa protein is either not present or not significantly phosphorylated in the absence of the factor. Other experiments showing that this *in vitro* kinase activity has the same sensitivity to staurosporine as that observed upon addition of CNTF to intact cells suggested that this associated tyrosine kinase activity is relevant to that which is required in the cell to mediate CNTF-induced responses. Furthermore, the 130 kDa protein appears to be a good candidate for this kinase since lysis of the cells in NP-40 does not give co-purification of either the 130 kDa protein or tyrosine kinase activity (not shown).

Paragraph beginning on page 129, line 6:

Experiments using specific antisera raised against portions of Jak1, Jak2, or Tyk2 reveal that all 3 of these kinases can become tyrosine phosphorylated following stimulation by CNTF, LIF, OSM, and IL6. [Figure 11A shows that] CNTF induces tyrosine phosphorylation of both Jak1 and Jak2 in EW1 cells, and these proteins appear to co-migrate with 130 and 131 kDa proteins that co-purify with the receptor complex immunoprecipitated with a-LIFRβ. Furthermore, the addition of IL6 + sIL6Rα [(Figure 11B)], as well as LIF and OSM (not shown) to EW-1 calls also results in phosphorylation of Jak1 and Jak2 but not Tyk2. In contrast, IL6 stimulated U266 cells give tyrosine phosphorylation of Tyk2 and Jak1 without apparent change in the phosphorylation status of Jak2. OSM treated SK-MES cells reveal tyrosine phosphorylation of primarily Jak2, with smaller changes in Tyk2 and Jak1. In each of these cases, tyrosine phosphorylation of the Jaks or Tyk2 is associated with an increase in their in vitro tyrosine kinase activity (not shown). These results stand in contrast to previous results showing that stimulation with GM-CSF, EPO, G-CSF, IFN-γ, or IL-3 only result in tyrosine phosphorylation of Jak2 ((Argetsinger *et al.*, *Cell 74*:237-244 (1993); Silvennoinen *et al.*, *Proc. Natl. Acad. Sci. USA* (in press;1993); Witthuhn *et al.*, *Cell 74*:227-236 (1993)). We conclude from these experiments that the CNTF family of factors can activate Jak1, Jak2, and Tyk2, although there is some variability in which Jak/Tyk family member is activated in a particular cell.

Paragraph beginning on page 130, line 2:

Transient transfections in COS cells were used to determine whether the Jaks could associate with the β receptor components in the absence of factors. These experiments used carboxyl terminally epitope-tagged versions of LIFRβ containing the 10 amino acid portion of c-myc that is recognized by the monoclonal antibody 9E10 (Davis *et al.*, *Science 253*:59-63 (1991)). COS cells were co-transfected with appropriate expression vectors encoding full length versions of LIFRβ and Jak1 or Jak2, and Brij 96 lysates were immunoprecipitated with 9E10 and then blotted with the antisera against either Jak1 or Jak2 [(Figure 12)]. These experiments show that either Jak can associate with LIFRβ in the absence of any added ligand. Furthermore, a truncated version of LIFRβ which retains only the first 76 amino acids of the cytoplasmic domain is fully capable of binding to Jak1 and Jak2 as well. This implicates the membrane proximal region of LIFRβ as the Jak binding domain, which is consistent with the homology between this region of the receptor with those in gp130 and EPOR that have been shown to be required for signal transduction upon factor binding (Murakami *et al.*, *Science 260*:11349-11353 (1991); Witthuhn *et al.*, *Cell 74*:227-236 (1993)).

Paragraph beginning on page 130, line 21:

Further experiments in COS cells were undertaken to establish whether co-transfection of the receptor β-components with the Jaks could reconstruct a ligand-induced functional response. Epitope-tagged gp130FLAG and IL6 were chosen for these

experiments, since gp130 homodimerizes and becomes tyrosine phosphorylated in response to IL6 + soluble IL6Rα, obviating the need for co-transfection with LIFRβ (Murakami *et al.*, *Proc. Natl. Acad. Sci. USA 88*:11349-11353 (1993); Davis *et al.*, *Science 260*:1805-1808 (1993)). Following stimulation with IL6 + sIL6Rα, neither mock transfected [(lane 1)] nor gp130FLAG transfected COS cells [(lanes 2-3)] revealed substantial tyrosine phosphorylation of gp130 following immunoprecipitation with anti-FLAG and α-PTyr immunoblotting [(Figure 13)]. In contrast, co-transfection with either Jak1 [(lanes 4-5)], Jak2 [(lanes 6-7)], or both Jak1 and Jak2 [(lanes 8-9)] gives rise to a substantial increase in the induced tyrosine phosphorylation of gp130 upon stimulation with IL6 + sIL6Rα.

Claim 43 have been amended as follows:

43. (Once amended) An isolated DNA molecule comprising a DNA sequence encoding at least 400 amino acids of a JAK3 peptide of SEQ ID NO: 16 [a Jak kinase peptide]; wherein said peptide binds a receptor for a cytokine selected from the group consisting of: IL-2, IL-3, IL-4, IL-5, IL-6, IL-7, IL-9, IL-11, OSM, LIF, G-CSF, EPO, IFN-γ and GM-CSF [having cytokine receptor binding activity].

COPY

FORM PTO-1449 INFORMATION DISCLOSURE STATEMENT	ATTY. DOCKET NO. 0656.0370004	APPLICATION NO. 09/397,967
	APPLICANT IHLE *et al.*	
	FILING DATE September 17, 1999	GROUP 1632

U.S. PATENT DOCUMENTS

EXAMINER INITIAL		DOCUMENT NUMBER	DATE	NAME	CLASS	SUB-CLASS	FILING DATE
QN	AA1	5,190,931	03/02/1993	Inouye *et al.*	435	91	11/15/1989
QN	AB1	6,210,654 B1	04/03/2001	Ihle *et al.*	424	9.2	10/08/1997
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EXAMINER INITIAL		DOCUMENT NUMBER	DATE	COUNTRY	CLASS	SUB-CLASS	TRANSLATION
QN	AL1	WO 92/10519	06/25/1992	WIPO			Yes No
	AM						Yes No
	AN						Yes No
	AO						Yes No
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QN	AR	1	Argetsinger, L.S. *et al.*, "Identification of JAK2 as a Growth Hormone Receptor-Associated Tyrosine Kinase," *Cell 74*:237-244, Cell Press (July 1993).
QN	AS	1	Bartholomew, C. and J.N. Ihle, "Retroviral Insertions 90-Kilobases Proximal to the *Evi-1* Myeloid Transforming Gene Activate Transcription from the Normal Promoter," *Mol. Cell. Biol. 11(4)*:1820-1828, American Society for Microbiology (1991).
QN	AT	1	Bird, T.A. *et al.*, "Evidence that MAP (Mitogen-Activated Protein) Kinase Activation May Be a Necessary but not Sufficient Signal for a Restricted Subset of Responses in IL-1 Treated Epidermoid Cells," *Cytokine 4(6)*:429-440, International Cytokine Society, Academic Press Ltd. (November 1992).

EXAMINER	DATE CONSIDERED
Quan Ngo	5/10/02

EXAMINER: Initial if reference considered, whether or not citation is in conformance with MPEP 609. Draw line through citation if not in conformance and not considered. Include copy of this form with next communication to Applicant.

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QN	AS	2	Carroll, M.P. *et al.*, "Erythropoietin Induces Raf-1 Activation and Raf-1 is Required for Erythropoietin-mediated Proliferation," *J. Biol. Chem. 266(23)*:14964-14969, The American Society for Biochemistry and Molecular Biology, Inc. (1991).
QN	AT	2	Carroll, M.P. *et al.*, "Interleukin-3 and Granulocyte-Macrophage Colony-stimulating Factor Mediate Rapid Phosphorylation and Activation of Cytosolic c-raf," *J. Biol. Chem. 265(32)*:19812-19817, The American Society for Biochemistry and Molecular Biology, Inc. (1990).

EXAMINER Quang Dang	DATE CONSIDERED 5/10/02

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QW	AR	3	Cleveland, J.L. *et al.*, "Tyrosine Kinase Oncogenes Abrogate Interleukin-3 Dependence of Murine Myeloid Cells through Signaling Pathways Involving *c-myc*: Conditional Regulation of *c-myc* Transcription by Temperature-Sensitive v-*abl*," *Mol. Cell. Biol. 9(12)*:5685-5695, American Society (1989).
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EXAMINER Quang Nguyen DATE CONSIDERED 5/10/02

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QW	AS	4	Fu, X.-Y., "A Transcription Factor with SH2 and SH3 Domains is Directly Activated by an Interferon α-Induced Cytoplasmic Protein Tyrosine Kinase(s)," *Cell 70*:323-335, Cell Press (July 1992).
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EXAMINER [signature] DATE CONSIDERED 6/10/02

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EXAMINER	DATE CONSIDERED
Amy Nguyen	5/10/02

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EXAMINER	DATE CONSIDERED
Quan Zhou	5/10/02

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	AO						Yes No
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EXAMINER INITIAL		DOCUMENT NUMBER	DATE	COUNTRY	CLASS	SUB-CLASS	TRANSLATION
	AL						Yes No
	AM						Yes No
	AN						Yes No
	AO						Yes No
	AP						Yes No

OTHER (Including Author, Title, Date, Pertinent Pages, etc.)

QW	AR	9	Isfort, R.J. *et al.*, "Interleukin 3 binds to a 140-kDa phosphotyrosine-containing cell surface protein," *Proc. Natl. Acad. Sci. USA 85*:7982-7986, National Academy of Sciences of the USA (1988).
QW	AS	9	Kappel, C.A. *et al.*, "Regulating gene expression in transgenic animals," *Curr. Opin. Biotech. 3*:548-553, Current Biology Ltd. (October 1992).
QW	AT	9	Koch, C.A. *et al.*, "SH2 and SH3 Domains: Elements That Control Interactions of Cytoplasmic Signaling Proteins," *Science 252*:668-674, Association for the Advancement of Science (1991).

EXAMINER	DATE CONSIDERED
Quang Nguyen	5/10/02

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FORM PTO-1449 INFORMATION DISCLOSURE STATEMENT	ATTY. DOCKET NO. 0656.0370004	APPLICATION NO. 09/397,967
	APPLICANT IHLE *et al.*	
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	AM						Yes No
	AN						Yes No
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	AP						Yes No

OTHER (Including Author, Title, Date, Pertinent Pages, etc.)

QW	AR	10	Linnekin, D. *et al.*, "Association of the erythropoietin receptor with protein tyrosine kinase activity," *Proc. Natl. Acad. Sci. USA 89*:6237-6241, National Academy of Sciences of the USA (July 1992).
QW	AS	10	Linnekin, D. and W.L. Farrar, "Signal transduction of human interleukin 3 and granulocyte-macrophage colony-stimulating factor through serine and tyrosine phosphorylation," *Biochem. J. 271*:317-324, London Portland Press On Behalf Of The Biochemical Society (1990).
QW	AT	10	Maliszewski, C.R. and W.C. Fanslow, "Soluble receptors for IL-1 and IL-4: biological activity and therapeutic potential," *TibTech 8*:324-329, Elsevier Science Publishers Ltd (1990).

EXAMINER Quan Zhou DATE CONSIDERED 6/10/02

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QW	AR	11	Mano, H. *et al.*, "Expression of a novel form of *Tec* kinase in hematopoietic cells and mapping of the gene to chromosome 5 near *Kit*," *Oncogene 8*:417-424, Macmillan Press Ltd. (February 1993).
QW	AS	11	Manthorpe, M. *et al.*, "Cholinergic Neuronotrophic Factors: Fractionation Properties of an Extract from Selected Chick Embryonic Eye Tissues," *J. Neurochem. 34(1)*:69-75, Blackwell Science Ltd. (1980).
QW	AT	11	Metcalf, D., "The molecular control of cell division, differentiation commitment and maturation in haemopoietic cells," *Nature 339*:27-30, Macmillan Publishers Ltd. (1989)

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QW	AR	12	Meydan, N. *et al.*, "Inhibition of acute lymphoblastic leukaemia by a Jak-2 inhibitor," *Nature 379*:645-648, Macmillan Publishers Ltd (February 1996).
QW	AS	12	Miura, O. *et al.*, "Inactivation of Erythropoietin Receptor Function by Point Mutations in a Region Having Homology with Other Cytokine Receptors," *Mol. Cell. Biol. 13(3)*:1788-1795, American Society for Microbiology (March 1993).
QW	AT	12	Miura, O. *et al.*, "Induction of Tyrosine Phosphorylation by the Erythropoietin Receptor Correlates with Mitogenesis," *Mol. Cell. Biol. 11(10)*:4895-4902, American Society for Microbiology (1991).

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DW	AR	13	Miyajima, A. *et al.*, "Cytokine Receptors and Signal Transduction," *Annu. Rev. Immunol. 10*:295-331, Annual Reviews Inc. (April 1992).
DW	AS	13	Morla, A.O. *et al.*, "Hematopoietic Growth Factors Activate the Tyrosine Phosphorylation of Distinct Sets of Proteins in Interleukin-3-Dependent Murine Cell Lines," *Mol. Cell. Biol. 8(5)*:2214-2218, American Society for Microbiology (1988).
DW	AT	13	Müller, M. *et al.*, "The protein tyrosine kinase JAK1 complements defects in interferon-α/β and -γ signal transduction," *Nature 366*:129-135, Macmillan Publishers Ltd (November 1993).

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	AR	14	O'Dell, T.J. *et al.*, "Long-term potentiation in the hippocampus is blocked by tyrosine kinase inhibitors," *Nature 353*:558-560, Macmillan Publishers Ltd (1991).
	AS	14	Ohtsuka, M. *et al.*, "Ligand-Induced Phosphorylation of the Colony-Stimulating Factor 1 Receptor Can Occur through an Intermolecular Reaction That Triggers Receptor Down Modulation," *Mol. Cell. Biol. 10(4)*:1664-1671, American Society for Microbiology (1990).
	AT	14	Olsson, T., "Cytokines in neuroinflammatory disease: role of myelin autoreactive T cell production of interferon-gamma," *J. Neuroimmunol. 40*:211-218, Elsevier Science Publishers B.V. (October 1992).

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RW	AR	15	Partanen, J. *et al.*, "Putative tyrosine kinases expressed in K-562 human leukemia cells," *Proc. Natl. Acad. Sci. USA 87*:8913-8917, National Academy of Sciences of the USA (1990).
RW	AS	15	Pellegrini, S. and C. Schindler, "Early events in signaling by interferons," *TIBS 18*:338-342, Elsevier Science Publishers (September 1993).
RW	AT	15	Pellegrini, S. *et al.*, "Use of a Selectable Marker Regulated by Alpha Interferon To Obtain Mutations in the Signaling Pathway," *Mol. Cell. Biol. 9(11)*:4605-4612, American Society for Microbiology (1989).

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QW	AR	16	Pritchard, M.A. *et al.*, "Two members of the JAK family of protein tyrosine kinases map to Chromosome 1p31.3 and 9p24," *Mammalian Genome 3*:36-38, Springer-Verlag (February 1992).
QW	AS	16	Quelle, F.W. *et al.*, "Interleukin 3, Granulocyte-Macrophage Colony-stimulating Factor, and Transfected Erythropoietin Receptors Mediate Tyrosine Phosphorylation of a Common Cytosolic Protein (pp100) in FDC-ER Cells," *J. Biol. Chem. 267(24)*:17055-17060, The American Society for Biochemistry and Molecular Biology, Inc. (August 1992).
QW	AT	16	Quelle, F.W. and D.M. Wojchowski, "Proliferative Action of Erythropoietin is Associated with Rapid Protein Tyrosine Phosphorylation in Responsive B6SUt.EP Cells," *J. Biol. Chem. 266(1)*:609-614, American Society for Biochemistry and Molecular Biology, Inc. (1991).

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QW	AR	17	Riordan, M.L. and J.C. Martin, "Oligonucleotide-based therapeutics," *Nature 350*:442-443, Macmillan Publishers Ltd. (April 1991).
QW	AS	17	Sanderson, C.J., "Interleukin-5, Eosinophils, and Disease," *Blood 79*:3101-3109, The American Society of Hematology (June 1992).
QW	AT	17	Schindler, C. *et al.*, "Interferon-Dependent Tyrosine Phosphorylation of a Latent Cytoplasmic Transcription Factor," *Science 257*:809-813, Association for the Advancement of Science (August 1992).

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	AO						Yes No
	AP						Yes No

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BW	AR	18	Schindler, C. *et al.*, "Proteins of transcription factor ISGF-3: One gene encodes the 91- and 84-kDa ISGF-3 proteins that are activated by interferon α," *Proc. Natl. Acad. Sci. USA 89*:7836-7839, National Academy of Sciences of the USA (August 1992).
BW	AS	18	Shohat, O. *et al.*, "Inhibition of Cell Growth Mediated by Plasmids Encoding p53 Anti-Sense," *Oncogene 1*:277-283, Stockton Press (1987).
BW	AT	18	Shuai, K. *et al.*, "Activation of Transcription by IFN-γ: Tyrosine Phosphorylation of a 91-kD DNA Binding Protein," *Science 258*:1808-1812, Association for the Advancement of Science (December 1992).

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	AM						Yes No
	AN						Yes No
	AO						Yes No
	AP						Yes No

OTHER (Including Author, Title, Date, Pertinent Pages, etc.)

EW	AR	19	Silvennoinen, O. *et al.*, "Structure of the murine Jak2 protein-tyrosine kinase and its role in interleukin 3 signal transduction," *Proc. Natl. Acad. Sci. USA 90*:8429-8433, National Academy of Sciences of the USA (September 1993).
EW	AS	19	Sorensen, P. *et al.*, "Interleukin-3 Stimulates the Tyrosine Phosphorylation of the 140-Kilodalton Interleukin-3 Receptor," *J. Biol. Chem. 264(32)*:19253-19258, American Society for Biochemistry and Molecular Biology, Inc. (1989).
EW	AT	19	Spangler, R. *et al.*, "Erythropoietin Increases *c-myc* mRNA by a Protein Kinase C-dependent Pathway," *J. Biol. Chem. 266(2)*:681-684, American Society for Biochemistry and Molecular Biology, Inc. (1991).

EXAMINER: EW DATE CONSIDERED: 5/10/02

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	AN						Yes No
	AO						Yes No
	AP						Yes No

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QW	AR	20	Stahl, N. *et al.*, "Cross-linking Identifies Leukemia Inhibitory Factor-binding Protein as a Ciliary Neurotrophic Factor Receptor Component," *J. Biol. Chem. 268(11)*:7628-7631, American Society for Biochemistry and Molecular Biology, Inc. (April 1993).
QW	AS	20	Takahashi, T. and T. Shirasawa, "Molecular Cloning of Rat JAK3, a Novel Member of the JAK Family of Protein Tyrosine Kinases," *FEBS Letts. 342*:124-128, Elsevier Science Publishers B.V. (March 1994).
QW	AT	20	Tepper, R.I. *et al.*, "IL-4 Induces Allergic-like Inflammatory Disease and Alters T Cell Development in Transgenic Mice," *Cell 62*:457-467, Cell Press (1990).

EXAMINER QW DATE CONSIDERED 5/16/02

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QW	AR	21	Torigoe, T. *et al.*, "Interleukin-3 Regulates the Activity of the LYN Protein-Tyrosine Kinase in Myeloid-Committed Leukemic Cell Lines," *Blood 80(3)*:617-624, W. B. Saunders (August 1992).
QW	AS	21	Turner, B. *et al.*, "Interleukin 2 induces tyrosine phosphorylation and activation of p72-74 Raf-1 kinase in a T-cell line," *Proc. Natl. Acad. Sci. USA 88*:1227-1231, National Academy of Sciences of the USA (1991).
QW	AT	21	Ullrich, A. and J. Schlessinger, "Signal Transduction by Receptors with Tyrosine Kinase Activity," *Cell 61*:203-212, MIT Press (1990).

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QW	AR	22	Velasquez, L. *et al.*, "A Protein Kinase in the Interferon α/β Signaling Pathway," *Cell 70*:313-322, MIT Press (July 1992).
QW	AS	22	Waldmann, T.A., "The IL-2/IL-2 receptor system: a target for rational immune intervention," *TiPS 14*:159-164, Elsevier Science Publishers Ltd (May 1993).
QW	AT	22	Wang, X. *et al.*, "Growth Hormone-promoted Tyrosyl Phosphorylation of a 121-kDa Growth Hormone Receptor-associated Protein," *J. Biol. Chem. 268(5)*:3573-3579, American Society for Biochemistry and Molecular Biology, Inc. (February 1993).

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QW	AR	23	Wang, Y. and G.M. Fuller, "Phosphorylation and Internalization of gp130 Occur After IL-6 Activation of Jak2 Kinase in Hepatocytes," *Molecular Biology of the Cell* 5:819-828, American Society for Cell Biology (July 1994).
QW	AS	23	Wilks, A.F. , "Two putative protein-tyrosine kinases identified by application of the polymerase chain reaction," *Proc. Natl. Acad. Sci. USA 86*: 1603-1607, National Academy of Sciences of the USA (1989).
QW	AT	23	Wilks, A.F., "Structure and Function of the Protein Tyrosine Kinases," *Prog. Growth Factor Res.* 2:97-111, Pergamon Press plc (1990).

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	AP						Yes No

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QW	AR	24	Wilks, A.F., "Cloning Members of Protein-Tyrosine Kinase Family Using Polymerase Chain Reaction," *Meth. Enzymol. 200*:533-546, Academic Press, Inc. (1991).
QW	AS	24	Wilks, A.F. *et al.*, "Two Novel Protein-Tyrosine Kinases, Each with a Second Phosphotransferase-Related Catalytic Domain, Define a New Class of Protein Kinase," *Mol. Cell. Biol. 11(4)*:2057-2065, American Society for Microbiology (1991).
QW	AT	24	Wilks, A.F. and A.G. Harpur, "Cytokine Signal Transduction and the JAK Family of Protein Tyrosine Kinases," *BioEssays 16(5)*:313-320, John Wiley & Sons, Inc. (May 1994).

EXAMINER	DATE CONSIDERED
	6/10/02

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FORM PTO-1449 INFORMATION DISCLOSURE STATEMENT	ATTY. DOCKET NO. 0656.0370004	APPLICATION NO. 09/397,967
	APPLICANT IHLE *et al.*	
	FILING DATE September 17, 1999	GROUP 1632

U.S. PATENT DOCUMENTS

EXAMINER INITIAL		DOCUMENT NUMBER	DATE	NAME	CLASS	SUB-CLASS	FILING DATE
	AA						
	AB						
	AC						
	AD						
	AE						
	AF						
	AG						
	AH						
	AI						
	AJ						
	AK						

FOREIGN PATENT DOCUMENTS

EXAMINER INITIAL		DOCUMENT NUMBER	DATE	COUNTRY	CLASS	SUB-CLASS	TRANSLATION
	AL						Yes No
	AM						Yes No
	AN						Yes No
	AO						Yes No
	AP						Yes No

OTHER (Including Author, Title, Date, Pertinent Pages, etc.)

QW	AR	25	Witthuhn, B.A. *et al.*, "JAK2 Associated with the Erythropoietin Receptor and Is Tyrosine Phosphorylated and Activated following Stimulation with Erythropoietin," *Cell 74*:227-236, MIT Press (July 1993).
QW	AS	25	Witthuhn, B.A. *et al.*, "Involvement of the Jak-3 Janus kinase in signalling by interleukins 2 and 4 in lymphoid and myelooid cells," *Nature 370*:153-157, Macmillan Publishers Ltd. (July 1994).
QW	AT	25	Yarden, Y. and A. Ullrich, "Growth Factor Receptor Tyrosine Kinases," *Ann. Rev. Biochem. 57*:443-478, Annual Reviews Inc. (1988)

EXAMINER QW DATE CONSIDERED 8/1/02

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	AL						Yes No
	AM						Yes No
	AN						Yes No
	AO						Yes No
	AP						Yes No

OTHER (Including Author, Title, Date, Pertinent Pages, etc.)

	AR	26	Yoshimura, A. and H.F. Lodish, "*In vitro* Phosphorylation of the Erythropoietin Receptor and an Associated Protein," *Mol. Cell. Biol. 12*:706-715, American Society for Microbiology (February 1992).
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EXAMINER

DATE CONSIDERED 6/10/02

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